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Ventilator Associated Events

Essay

Submitted for Partial Fulfillment of Master Degree

in Intensive Care

By

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2016

Acknowledgement

First of all, I wish to offer my deepest gratitude to **ALLAH** for enabling me to achieve this work.

I would like to express my highest gratitude to **Prof. Dr. Mohamed Ismael El Saidi,** Professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for giving me the privilege of working under his instructive and helpful guidance.

I am truly Indebted to **Dr. Ibrahim Mamdouh Esmat,** Lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for his generous help & endless advices.

And special thanks to **Dr. Hany Magdy Fahim,** Lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University for his great help and support throughout this work.

Mohamed Salah

List of Contents

List of Abbreviations

Abb.	Meaning
A/C	Assist-Control
ABG	Arterial blood gas
ACRF	Acute exacerbation of chronic respiratory failure
ALI	Acute lung injury
ARDS	Acute respiratory distress syndrome
ASV	Adaptive support ventilation
BAL	Bronchoalveolar lavage
BPAP	Bilevel Positive Airway Pressure
CDC	The Center for Disease Control and Prevention
CMV	Controlled Mechanical Ventilation
CPAP	Continuous Positive Airway Pressure
CPIS	Clinical Pulmonary Infection Score
CRP	C-reactive protein
DO_2	Oxygen delivery
ECMO	Extracorporeal membrane oxygenation
EPAP	Expiratory positive airway pressure
ETT	Endotracheal tube
FRC	Functional Residual Capacity
ICU	Intensive care unit
IPAP	Inspiratory positive airway pressure
ITP	Intrathoracic pressure
IVAC	Infection-related Ventilator-Associated Complication
MIP	Maximum inspiratory pressure
MRSA	Methicillin resistant Staphylococcus aureus
MSSA	Methicillin sensitive Staphylococcus aureus
MV	Mechanical ventilation
NAVA	Neurally adjusted ventilatory assist
NHSN	National Healthcare Safety Network
NICE	The National Institute for Health and Care Excellence

Abb.	Meaning
NIPPV	Non-invasive positive pressure ventilation
NIV	Non-invasive ventilation
ODC	Oxygen dissociation curve
$PaCO_2$	Arterial carbon dioxide tension
PAO_2	Alveolar O_2 tension
PaO_2	Arterial oxygen tension
PAV	Proportional assist ventilation
PEEP	Positive end-expiratory pressure
PPV	Positive-pressure mechanical ventilation
PSB	Protected specimen brush
PSV	Pressure Support Ventilation
PVAP	Possible and Probable VAP
PVR	Pulmonary vascular resistance
RF	Respiratory failure
ROS	Reactive O_2 species
SATs and SBTs	Spontaneous awakening and breathing trials
SIMV	Synchronized Intermittent Mandatory Ventilation
sTREM-1	Soluble triggering receptor expressed on myeloid cells-1
TEF	Formation of a tracheoesophageal fistula
VA/Q	Ventilation-perfusion ratio
VACs	Ventilator Associated Conditions
VAE	Ventilator-Associated Events
VAP	Ventilator-associated pneumonia
VC	Vital capacity
VILI	Ventilator-induced lung injury
V-PSV	Variable pressure support ventilation

List of Tables

List of Figures

Introduction

A mechanical ventilator is a machine that makes it easier for patients to breath until they are able to breathe completely on their own. Early mechanical ventilation in humans was described in the 18th century by Hunter, who performed ventilation using bellows to artificially ventilate drowned patients through a tracheostomy. In the same century, Kite described the technique of endotracheal intubation. After a period of negative pressure ventilation, induced by the invention of the iron lung in 1929, Ibsen finally introduced positive pressure ventilation outside the operating theatre in 1952. This development marked the birth of the modern intensive care unit (ICU).**(Puri et al., 2009)**

There are many clinical indications for mechanical ventilation but the primary indication is impending or existing respiratory failure despite maximal treatment.**(Esteban et al., 2002)**

However, many complications occurred by mechanical ventilation itself, such as barotrauma, volutrauma, atelectotrauma and biotrauma. Ventilator-induced lung injury (VILI) is major cause of death in the

acute respiratory distress syndrome (ARDS) with multiple organ failure together. Such complications can lead to longer duration of mechanical ventilation, longer stays in the ICU, increased healthcare costs, increased risk of disability and death.**(Plotz et al., 2004)**

Multiple lines of evidence, however, suggest that Ventilator Associated Conditions (VACs) are complications rather than just markers of severity of illness. the definition requires patients to have a period of at least 2 days of respiratory stability or improvement before they are eligible for VAC, published estimates of attributable length of stay and mortality were adjusted for baseline severity of illness and qualitative analysis of VAC suggest that most events are caused by potentially preventable conditions acquired in the ICU, namely, pneumonia, pulmonary edema, atelectasis and ARDS.**(Hayashi et al., 2013)**

There are many strategies that can be applied to minimize the risk of VILI. The most important is the use of lower tidal volumes for positive-pressure ventilation. Low tidal volumes can result in airway collapse, particularly at the end of expiration which can be prevented by adding positive end-expiratory pressure (PEEP) and by recruitment maneuvers. Another consequence of low volume

ventilation is a reduction in CO_2 elimination via the lungs leading to hypercapnia and respiratory acidosis. Allowing hypercapnia to persist in favor of maintaining lung-protective low-volume ventilation is known as permissive hypercapnia.**(Girad and Bernard, 2007)**

Aim of the Work

The aim of this essay is to highlight on the incidence, possible hazards, how to treat and methods to prevent complications of mechanical ventilation.

Respiratory Failure

The respiratory system performs the vital function of gaseous exchange. O_2 is transported through the upper airways to the alveoli that diffuses across the alveolocapillary membrane and enters the capillary blood. There, it combines with haemoglobin and is transported by the arterial blood to the tissues. In the tissues, the O_2 is utilized for adenosine triphosphate production which is essential for all metabolic processes. The major product of cellular metabolism, CO_2, diffuses from the tissues into the capillary blood, where a major portion of it is hydrated as carbonic acid and transported to the lungs by the venous blood. In the lungs, it diffuses from the pulmonary blood into the alveoli and is exhaled into the atmosphere (Expiration). Gaseous exchange appropriate to the metabolic demand is essential to maintain homeostasis.**(Neema, 2003)**

- **Physiology of respiration**

Respiration is accomplished and regulated by an intricate set of structures. These structures include: (1) the lungs that provide the gas exchange surface; (2) the conducting airways that convey the air into and out of the lungs; (3) the thoracic wall that acts as a bellows which

supports and protects the lungs; (4) the respiratory muscles that creates the energy necessary for the movement of air into and out of the lungs; and (5) the respiratory centers with their sensitive receptors and communicating nerves that control and regulate ventilation. Pathologic processes can affect any of these functional components. The interactions of cardiopulmonary, nervous and musculoskeletal systems can be disrupted by disease, by surgery and by anaesthetic agents.**(Papadakos, 2002)**

Gaseous exchange between the environment and the pulmonary capillary blood constitutes external respiration. The functioning unit of the lung is alveolus with its capillary network. Various factors govern transport of air from the environment to the alveoli (ventilation) and supply of blood to the pulmonary capillaries (perfusion). Henry's law dictates that when a solution is exposed to an atmosphere of gas an equilibration of partial pressures follow between the gas molecules dissolved in the liquid and the gas molecules in the atmosphere. Consequently, partial pressure of O_2 and CO_2 in the blood leaving the pulmonary capillaries (pulmonary venous blood) is equal to the partial pressure of O_2 and CO_2 achieved in the alveolus after equilibration.**(Neema, 2003)**

At equilibrium, the partial pressure of O_2 and CO_2 results from a dynamic equilibrium between O_2 delivery to the alveolus and O_2 extraction from the alveolus; and CO_2 delivery to the alveolus and CO_2 removal from the alveolus. Delivery of O_2 to the alveolus is directly related to the sweep rate of air (ventilation), and composition of the sweeping gas (partial pressure of O_2 in the inspiratory air FiO_2). In general, alveolar O_2 tension (PAO_2) increases with increase in inspiratory O_2 tension and increase in ventilation. Extraction of O_2 from the alveolus is determined by the saturation, quality and quantity of the haemoglobin of the blood perfusing the alveoli. The O_2 saturation of the haemoglobin in the pulmonary capillary blood is affected by the supply of O_2 to the tissues (cardiac output) and the extraction of the O_2 by the tissues (metabolism).**(Neema, 2003)**

Oxygen delivery (DO_2) is the rate at which oxygen is transported from the lungs to the microcirculation: DO_2 (mL/min) = Q x CaO_2 where Q is the cardiac output. Oxygen consumption (VO_2) is the rate at which oxygen is removed from the blood for use by the tissues. It can be measured directly or calculated. Calculation of VO_2 can be performed by rearranging the Fick equation: VO_2 (mL O_2/min) = Q x (CaO_2 - CvO_2).**(Neema, 2003)**